that these fragments, which would normally be small enough to be cleared by glomerular filtration, accumulate in the circulation of patients with end-stage renal disease and crossreact in immunoassays for cTnT.

Recent evidence, unavailable to Kanderian and Francis when their paper was accepted, argues against this. Firstly, cTnT concentrations are significantly increased among patients with CKD long before end stage is reached (i.e. when significant glomerular filtration remains).[3] Secondly, using a direct gel-filtration chromatography approach, we have shown that the form of cTnT circulating in dialysis patients and reacting in the commercial cTnT immunoassay is an intact, free form, identical in size to that observed among non-CKD patients following an acute coronary syndrome, with no evidence of smaller molecular weight fragments.[4] Diris *et al.*[2] used a complex analytical approach including the use of Western blotting, which is known to be susceptible to artifact.

Much work is needed before the pathophysiology underlying cardiac troponin increases in CKD is fully understood. However, it is important that this presentation is not dismissed as an artifact due to fragment accumulation: increases in CKD are real and predict death.

1. Kanderian AS, Francis GS. Cardiac troponins and chronic kidney disease. *Kidney Int* 2006; **69**: 1112–1114.
2. Diris JH, Hackeng CM, Kooman JP *et al.* Impaired renal clearance explains elevated troponin T fragments in hemodialysis patients. *Circulation* 2004; **109**: 23–25.
3. Abbas NA, John RI, Webb MC *et al.* Cardiac troponins and renal function in non-dialysis patients with chronic kidney disease. *Clin Chem* 2005; **51**: 2059–2066.
4. Fahie-Wilson MN, Carmichael DJ, Delaney MP *et al.* Fragments of cardiac troponin T are not responsible for the elevated serum concentrations observed in patients with kidney failure. *Clin Chem* 2006; **52**: 414–420.

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Response to 'Cardiac troponins and chronic kidney disease'

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We thank Lamb *et al.*[1] for bringing to our attention new data regarding the mechanism of elevated circulating troponin levels (cTnT) in patients with renal dysfunction. We were unaware of their recently published results. Their work indicates that it is the free-form cTnT that is being measured in patients with chronic kidney disease (CKD), identical in size to that of cTnT observed in non-renal disease patients, and not a smaller fragment of cTnT as suggested earlier by Diris *et al.*[2] We agree that although the mechanism of altered cTnT levels in CKD is not fully understood, increased cTnT levels are consistently associated with an incremental change in morbidity and mortality, and cannot be dismissed as laboratory artifact.

1. Fahie-Wilson MN, Carmichael DJ, Delaney MP *et al.* Fragments of cardiac troponin T are not responsible for the elevated serum concentrations observed in patients with kidney failure. *Clin Chem* 2006; **52**: 414–420.
2. Diris JH, Hackeng CM, Kooman JP *et al.* Impaired renal clearance explains elevated troponin T fragments in hemodialysis patients. *Circulation* 2004; **109**: 23–25.

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Differences between type I and II membranoproliferative glomerulonephritis

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To the Editor: Little *et al.*[1] recently analyzed post-transplantation recurrence risk in patients with membranoproliferative glomerulonephritis (MPGN). They found that age and crescents on initial biopsy determined this risk rather than the type of MPGN. The authors suggest that type II MPGN may not be very different from type I MPGN. However, it is evident that type I and II MPGN are pathologically and pathogenetically different entities. Likewise, membranous nephropathy and focal segmental glomerulosclerosis are totally different diseases, although the nephrotic syndrome rather than the diagnosis determines outcome. Crescents may be a common risk factor for recurrent disease, as has been suggested for immunoglobulin A-nephropathy.[2] From the data we cannot retrieve if clinically silent recurrences of type II MPGN have been missed. Furthermore, the multivariate analysis did not include potential risk factors as repeated transplantation and living-related donor transplantation.[3] In our single-center studies, we noted significant differences between type I and II MPGN with regard to post-transplant recurrence. A recurrence of type I MPGN occurred in almost 50% of recipients and was invariably accompanied by clinically significant proteinuria. Increased risk of recurrence was observed with human lymphocyte antigen-identical living-related donor kidneys, the human lymphocyte antigen-B8DR3 haplotype, and repeated transplants.[3] In contrast, all patients with type II MPGN who had been biopsied (11 of 13) showed a recurrence. Most recurrences were, however, clinically silent and required immunofluorescence or electron microscopy for diagnosis. Still, patients with type II MPGN had poor graft survival. Crescents in the original biopsy were not specific predictors.[4]